Negative Life Events, Anxiety, Depression and Coping Ability (Stress) as Related to Chronic Periodontitis

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In animal models it has been shown experimentally that psychological stress may negatively affect the outcome of periodontitis. Several studies in various populations have confirmed an association between negative life events, the level of depression and anxiety, as well as personality traits and periodontitis. The aim of the present study was to explore these relationships in a Norwegian adult population. Patients aged 40+ yr were selected on the basis of their periodontitis experience as assessed radiographically, and the participants were grouped in 2 groups: group 1 consisted of periodontally healthy subjects (no detectable bone loss); and group 2 consisted of subjects with >3mm radiographic bone loss in >3 sites. Four different questionnaires were employed to assess the participants' level of anxiety, depression and neuroticism, as well as their Health Locus of Control. Negative life events and smoking habits were also recorded. The results revealed a statistically significant bivariate relationship between periodontitis and negative life events or the degree of anxiety among the selected psychosocial variables. However, these associations did not appear to be significant when smoking was introduced in a linear multiple regression model. One explanation offered is that good access to dental care and a high utilization of oral health services in Norway may have compensated for the possible underlying causal effects of psychosocial stressors on periodontal health. Thus, comparisons of such studies between different populations may not always be feasible.

Key words: psychological stress, periodontitis, epidemiology

The psychological response of the organism to a perceived challenge or threat is referred to as stress. The stress response is essential for the survival of the organism and should not be regarded as undesirable (Breivik et al, 1996). The function of the stress response is to prepare the organism to meet the challenge or threat appropriately, and is therefore homeostatic. Stress becomes dangerous for the organism only when it is sustained or when one or more aspects of the neuroendocrine response act on an organ system which, for some reason, is already predisposed to pathology. The immune system is bidirectionally linked to the psyche, the nervous system and the endocrine system, and the brain areas involved in the classical stress response regulate the immune response to bacterial and other antigenic challenges (Ader et al, 1995; Chrousos, 1995).

Periodontitis is a multifactorial disorder initiated by bacteria and is believed to be the result of disrupted host-parasite equilibrium resulting in tissue destruction (Offenbacher, 1996). Psychological stress has been suggested as a disruptive factor in the homeostasis of oral bacteria and the host's immune system. We may expect different outcomes of periodontal disease depending on the interplay of virulent bacteria, host inflammatory responses, stress experiences of the individual (trait characteristics), and stress characteristics of his/her current situation (state characteristics) (Axtelius et al, 1997). A variety of psychological factors including mood, personality characteristics, coping style, suppressed anger, a sense of helplessness and defensiveness may all affect the way a person deals with emotional stress. It is primarily the state of learned helplessness which seems to alter the neuroendocrine

and immune system in a negative direction. Thus, the sense of controlling stressful experiences (coping ability) may modify immune responses affected by stress (Kavelaars et al, 1999).

There is a relatively consistent literature suggesting a relationship between stressful life events and increased risk of various pathological conditions, including infectious diseases (McClelland et al, 1980; Cohen and Williamson, 1991; Cohen et al, 1993; Ursin and Olff, 1993; Sheridan et al, 1994). Also, the relationship between stress and periodontal disease has been reported (Monteiro da Silva et al, 1996; Moss et al, 1996; Genco et al, 1998; Hugoson et al, 2002; Wimmer et al, 2002). Although the microbial impact on acute necrotizing ulcerative gingivitis (ANUG) is crucial, it has long been recognized that psychological stress affects the development of the disease. Indications that the central nervous system may regulate immune functions originate from animal studies showing that damage to particular brain areas causes altered immune system responses (Breivik et al, 2002).

Physiological responses to emotional stressors have been shown to modulate the immune system in at least 3 different ways: through the autonomic nervous system pathway; through the release of hypothalamic and pituitary hormones; and through the release of neuropeptides (Kiecolt-Glaser and Glaser, 1995; Ader et al, 1990; Blalock, 1994).

The aim of the present study was to explore the associations between psychological stress components and chronic periodontitis in a Norwegian adult population.

MATERIAL AND METHODS

Patient Selection

Dental patients seeking care at the Faculty of Dentistry, University of Oslo and at a private practice in Ålesund, Norway during 1996–2000 were recruited for this study. The participants were aged 40+ yr with at least 20 teeth displaying either radiographic bone loss >3 mm in >3 sites (periodontitis group), or no sites with radiographic bone loss >1 mm (control group). The periodontitis group and the control group were balanced for age and gender separately in both locations. Radiographic bone loss was regarded as an expression of accumulated exposure to periodontitis. Full mouth X-rays were assessed for bone loss by one trained examiner using an ADA Realist® X-ray viewer with approximately 10 times magnification. Recordings were made with a drawing compass and adjusted to the exact magnification of the viewer (Hansen et al, 1984).

Patients with systematic disease and/or on medication related to periodontitis were excluded. A total of 128 patients were invited to participate; 96 accepted and completed the study, yielding a response rate of 75%. The response rate for the control group and the test group was 74% and 77%, respectively. There were no significant differences concerning age and gender among patients recruited from the 2 locations (Table 1), nor were there any such differences between the periodontitis group and the healthy group (Table 2).

Psychological Measures

Four different questionnaires were employed and the patients replied to the questionnaires in the presence of an examiner, who assisted in the interpretation of the questions as appropriate:

- The Hospital Anxiety (HAD_1) Depression (HAD_2) Scale comprises 14 questions related to reactions (joy, events and literature), mood (humor/appearance) and retardation, and is supposed to reflect states of anxiety and depression (Malt et al, 1997).
- The Eysenck Personality Questionnaire (EPQ-N) represents a personality scale of neuroticism. The typical high EPQ-N scorer is an anxious worrying individual, moody, frequently depressed, and likely to suffer from various psychosomatic disorders (Grayson, 1986; Blomhoff and Malt, 1995; Eysenck and Eysenck, 1997).
- The Multidimensional Health Locus of Control Scale (short version) assesses control beliefs relevant to health, building upon Levenson's dimensions of personal control (internal (HLC-i)), the effectiveness of powerful others (external (HLC-e)), and the role of chance (HLC-c) in determining one's own health status (Regis et al, 1994).
- Negative life events (NLE), e.g. loss of relatives (mainly during childhood) were recorded.

Table 1 Mean age and standard deviation (S.D.) of participants from the 2 locations according to gender

	N	Males	N	Females	N	Total
Oslo	16	59.9 (11.1)	14	62.3 (11.5)	30	61.0 (11.2)
Ålesund	32	53.8 (9.0)	34	56.7 (9.5)	66	55.3 (9.3)
Total	48	55.8 (10.0)	48	58.3 (10.4)	96	57.1 (10.2)

Table 2 Distribution of participants in test and control groups according to age and gender

		Mean age (S.D.)		
Groups Range	N	Males	N	Females
Periodontitis	22	54.2 (8.1)	20	54.8 (9.3)
No periodontitis	26	57.2 (11.4)	28	60.9 (10.5)
Total	48	55.8 (10.0)	48	58.3 (10.4)

In addition, smoking habits were recorded. In order to provide a continuous measure for the individual's cigarette consumption an index expressing the total smoking exposure (TSE) was calculated according to the formula: *A x 360 x B*, where A is the number of cigarettes per day and B is number of years smoking. For those who had stopped smoking <10 years ago, the total smoking experience was reduced by 10% per year since stopping.
Also the participants were asked to assess their subjective opinion of their own general health status on a 5-point scale, which was collapsed to a 3-point scale before analyses.

Statistical Methods

The 4 psychosocial indices were organized as additive indices on an ordinal scale and the strength of the associations among the indices was expressed by Spearman's rho. Differences in mean index values according to periodontal status were calculated and tested for intergroup significance by means of student's t-test. In order to test for differences in distribution of periodontally healthy individuals and periodontally diseased persons according to the index values, the indices were collapsed to high and low using the median as the cut-off point. Smoking status was assessed as non-smokers (smokers who had quit smoking ≥10 years ago were regarded as non-smokers), previous smokers, and current smokers. Tests for significance were carried out using $\chi 2$ statistics. The relative influences of the various variables upon periodontal health and disease were assessed using a logistic multiple regression model.

RESULTS

The association among the various indices and periodontal status is shown in Table 3. The strongest significant associations were observed between periodontal disease and smoking (TSE) (Table 4). Weaker, but also significant bivariate associations were observed between smoking and anxiety, anxiety and depression, anxiety and neuroticism, depression and neuroticism, and depression and negative life events before the age of 15.

Table 3 The bivariate association (Spearman's rho) among various selected variables

	Perio status	Age	TSE	HAD_1	HAD_2	EPQ	HLC-i	HLC-e	HLC-c	NLE
Periodontal status	1.00	-.19	.58**	.18	.14	.12	-.05	.05	.05	.10
Age		1.00	-.07	.04	.11	.16	.01	.12	.02	.12
TSE			1.00	.24*	.11	.16	-.11	.02	.06	.07
HAD_1				1.00	.44**	.70**	-.02	.04	.04	.17
HAD_2					1.00	.49**	-.01	-.15	.08	.23*
EPQ						1.00	-.04	-.03	-.07	.18
HLC-i							1.00	.06	.04	-.07
HLC-e								1.00	.30**	-.13
HLC-c									1.00	.06
NLE										1.00

* p <0.05
** p <0.01
TSE = Total Smoking Exposure
HAD_1 = Hospital Anxiety
HAD_2 = Depression
EPQ = Eysenck Personality Questionnaire
HLC-i = Health Locus of Control-internal
HLC-e = Health Locus of Control-external
HLC-c = Health Locus of Control-chance
NLE = Negative Life Events

Table 4 The number of smokers, previous smokers and non-smokers related to the periodontitis group and the control group, respectively, and the Total Smoking Exposure (TSE) among smokers and previous smokers

Groups	Smokers	Previous smokers	Non-smokers	TSE*
Periodontitis	26	10	6	169659**
Control	10	3	41	13896**

TSE = Total Smoking Exposure (No. of cigarettes per day x 360 x years of smoking) (Reduced by 10% for each year since quitting for previous smokers)
** p <0.001 (students t-test)

There were statistically significant intergroup differences in age, total smoking exposure, and mean values of some of the psychosocial indices (anxiety and neuroticism) according to periodontal status (Table 5).
Chi-square tests revealed statistically significant differences in the distribution of subjects according to smoking habits, negative life events and anxiety, and periodontal status. No other variables yielded statistically significant differences in distribution according to periodontal status (Table 6; Fig. 1).
In a linear multivariate regression model, smoking was the only variable significantly associated with periodontal status.

Table 5 Mean (S.D.) of selected variables according to periodontal status

	N	Periodontitis	N	No periodontitis	p-value*
Age	42	54.48 (8.60)	54	59.11 (10.97)	0.031
TSE	42	145422 (107509)	54	33245 (67931)	0.003
HAD_1	42	6.03 (4.49)	54	4.24 (2.91)	0.005
HAD_2	42	3.83 (2.66)	53	3.24 (2.98)	0.394
EPQ	42	2.66 (2.83)	53	1.89 (2.03)	0.008
HLC-i	41	25.10 (6.08)	52	25.86 (5.59)	0.337
HLC-e	42	22.43 (6.36)	52	21.52 (6.51)	0.891
HLC-c	41	19.32 (5.50)	52	18.41 (6.30)	0.821

* Student's t-test
TSE = Total Smoking Exposure
HAD_1 = Hospital Anxiety
HAD_2 = Depression
EPQ = Eysenck Personality Questionnaire
HLC-i = Health Locus of Control-internal
HLC-e = Health Locus of Control-external
HLC-c = Health Locus of Control-chance

Table 6 Number of valid cases (N), χ^2-value, degrees of freedom (df) and p-value for various variables tested against periodontal status

Variables	N	χ^2-value	df	p-value
Present health	95	2.514	2	0.284
Smoking status	96	36.007	2	0.000
NLE	95	1.006	1	0.316
HAD_1	96	4.832	2	0.089
HAD_2	96	1.604	2	0.448
EPQ	92	3.208	2	0.201
HLC-i	93	1.691	2	0.429
HLC-e	94	0.794	2	0.672
HLC-c	91	0.495	2	0.781

NLE = Negative Life Events
HAD_1 = Hospital Anxiety
HAD_2 = Depression
EPQ = Eysenck Personality Questionnaire
HLC-i = Health Locus of Control-internal
HLC-e = Health Locus of Control-external
HLC-c = Health Locus of Control-chance

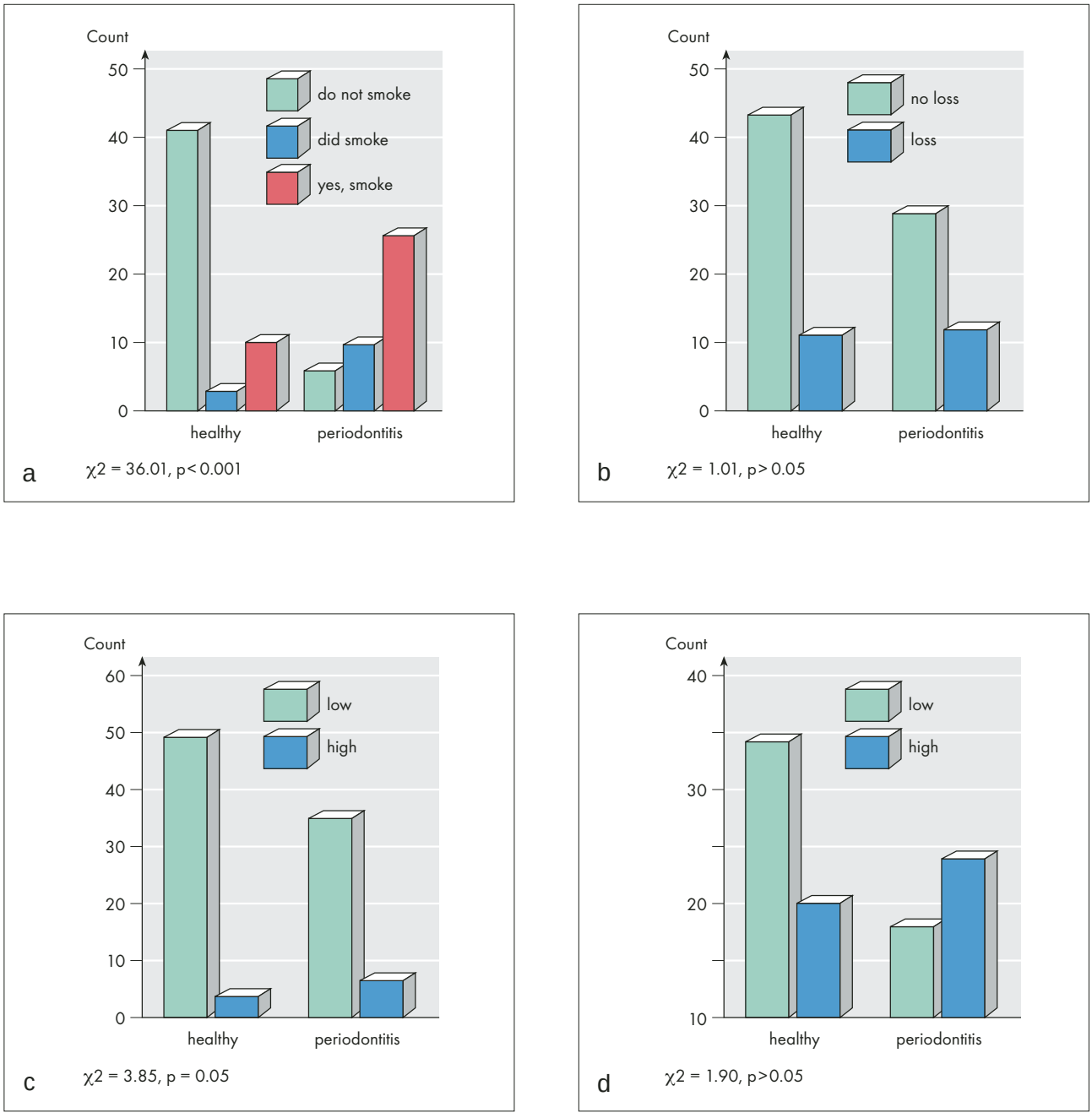

Fig. 1a to d Distribution of periodontally diseased and healthy subjects according to smoking status (a), loss experiences (negative life events) before the age of 15 (b), anxiety status (c) and level of depression (d) and the respective χ^2 and p values.

DISCUSSION

Several studies have reported associations between stress factors and gingivitis (Deinzer et al, 1998) and periodontitis (Green et al, 1986; Marcenes and Sheiham, 1992; Genco et al, 1998). In contrast to the previous studies we could only show a significant relationship for smoking habits when interactions between variables were accounted for in a multivariate regression model. This may indicate that stress factors will have different effects upon the consequences of chronic inflammations in various populations, depending on availability and consumption of oral health services, and cultural and/or social differences in coping with stress. Thus, it may be difficult to compare results from the various studies because the participants were from different geographical regions, cultural backgrounds and belonged to different social groups.

However, weak bivariate associations between periodontal disease status and some of the psychosocial variables were observed. When analyzed multivariately, inadequate sample size may be one reason for lack of significance of those factors; another reason may be that the variables employed did not detect variations sufficiently. Thus, we used a definition of periodontal disease that might have included too many cases of moderate disease, which would have been influenced by too many factors in addition to those included (e.g. oral hygiene or specific infections). Also, the grouping of patients was made on the basis of quantitative radiographic changes of marginal alveolar bone level that reflected the cumulative disease experience, without taking the degree of inflammation into account. In the present study, the 3 different questionnaires employed to investigate differences in psychological parameters are generally accepted to reveal the personality traits and reaction patterns under investigation.

Smoking exposure in the present study was strongly related to the radiographically recorded periodontitis experience. Since smoking, in addition to its effect on the course of periodontitis, is also correlated with some of the other variables in this study, it may have a strong confounding effect and thereby a tendency to overshadow the effects of other variables.

Thus, in the present study, any statistically significant association between an accumulated periodontal experience and various stress factors could not be demonstrated.

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